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Successful use of a modified Goeckerman regimen in the treatment of generalized prurigo nodularis

To the Editor: Prurigo nodularis (PN) is a severe morphologic manifestation of chronic pruritus. Although the exact pathogenesis is unknown, it is likely that neural sensitization to itch, inflammation, and trauma contribute to the evolution of PN.

Four patients with recalcitrant generalized PN were treated with an outpatient modified Goeckerman regimen 5 days per week at the University of California San Francisco between June 2013 and February 2014 (Table I). All patients had previously failed standard narrowband or broadband UVB therapy. The Goeckerman regimen consisted of daily multiple-step broadband UVB therapy followed by application of crude coal tar (CCT) and topical corticosteroids under occlusion for 4 hours each day. Patients were instructed to apply liquor carbonis detergens and topical corticosteroids at home following each session. Treatment was discontinued when satisfactory clinical response was achieved.

Patients underwent a mean 45.5 sessions of modified Goeckerman therapy, and a mean total broadband UVB dose of 11.8 J was delivered to the extremities, the body area receiving the highest UVB exposure. In patients 1, 2, and 4, pruritus resolved within the first 5 treatments and nodules flattened over the following weeks (Fig 1). Patient 3 was noted to be noncompliant with attendance and home therapy and demonstrated a more prolonged course of therapy. The only adverse events were mild phototoxic reactions that were easily managed by reduction of the broadband UVB dose.

Following completion of the modified Goeckerman regimen, patients were instructed to continue 3 sessions of broadband UVB phototherapy per week with topical steroids applied to recalcitrant nodules as needed. Patients 2 and 3 maintained clearance for 8 and 2.5 months before recurrence, respectively, while patients 1 and 4 remain in remission 5 months and 7 months after treatment at the time of writing.

It is increasingly recognized that neural sensitization to itch, neural proliferation, and neurogenic inflammation are primary components initiating and maintaining chronic pruritus. Nerve growth factor (NGF) and calcitonin gene-related peptide (CGRP) are central mediators implicated in these processes.[1-3] Furthermore, Th2 cell-derived IL-31 mediates pruritus through interaction with neural IL-31RA.[4] PN lesions demonstrate increased levels of NGF, an increased number of dermal CGRP-positive nerve fibers, and markedly increased levels of IL-31 mRNA.[4-6]

UVB radiation reduces levels of NGF and dermal CGRP-positive nerve fibers.[7] It also results in immunosuppressive effects, which may in turn reduce levels of IL-31.[8] However, all of our patients failed previous broadband or narrowband UVB therapy.

It is possible that topical CCT, which produces anti-inflammatory and antipruritic effects, increases the efficacy of UVB radiation in PN as demonstrated in psoriasis. Furthermore, occlusion enhances the anti-inflammatory and antipruritic effects of topical corticosteroids, and occlusion itself serves as a temporary physical barrier interrupting the itch-scratch cycle.

A modified Goeckerman regimen may be an effective treatment for PN by targeting both neurologic and inflammatory components of chronic itch. Based on the efficacy in these patients, dermatologists should consider Goeckerman therapy for patients with severe pruritus resulting in PN, even if they have failed standard phototherapy.

Eric Sorenson, AB,[a] Ethan Levin, MD,[b] John Koo, MD,[b] and Timothy G. Berger, MD[c]

Keck School of Medicine, University of Southern California, Los Angeles,[a] Psoriasis Treatment Center, University of California San Francisco[b]; and Department of Dermatology, University of California San Francisco[c]

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Table I. Patient characteristics and courses of therapy

Patient	Age	Location	Baseline pruritus	Duration of disease	Associated condition(s)	Previous therapy	Duration of Goeckerman therapy	Maximum daily broadband UVB dose	Topical regimen	Adverse events	Total broadband UVB dose	Outcome	Length of remission
1	66 years	Trunk, extremities, and scalp	Severe	1.5 years	Unknown	Clobetasol 0.05% lotion, hydroxyzine, cetirizine, isotretinoin, narrowband UVB	43 days	140 mJ to the scalp and trunk, 260 mJ to the extremities	2% coal tar, triamcinolone 0.1% ointment to the trunk and extremities, triamcinolone 0.1% solution to the scalp	1+ erythema	3260 mJ to the scalp and trunk, 4070 mJ to the extremities	Resolution of pruritus Flattening of nearly all nodules with a few recalcitrant lesions on the left arm	N/A
2	15 years	Trunk, extremities, and scalp	Severe	4 years	Unknown	Clobetasol 0.05% ointment, topical tacrolimus, broadband UVB, modified Goeckerman regimen	40 days	150 mJ to the scalp, 490 mJ to the trunk, 1040 mJ to the extremities	2%-10% coal tar, clobetasol 0.05% ointment to the trunk and extremities, triamcinolone 0.1% lotion or liquor carbonis detergens 20% in Nutraderm to the scalp	1+ erythema	3870 mJ to the scalp and trunk, 13,500 mJ to the extremities	Resolution of pruritus Complete flattening of nodules on the trunk with a few recalcitrant lesions on the extremities	8 months
3	68 years	Trunk, extremities, and scalp	Severe	21 years	Atopic dermatitis, hepatitis B	Clobetasol 0.05% ointment, hydroxyzine, broadband UVB, modified Goeckerman regimen	70 days	190 mJ to the scalp, 310 mJ to the trunk, 825 mJ to the extremities	2%-5% coal tar, clobetasol 0.05% ointment to the trunk and extremities transitioned to triamcinolone 0.1% ointment, clobetasol 0.05% solution to the scalp transitioned to liquor carbonis detergens 20% in Nutraderm	1+ erythema	1230 mJ to the scalp, 20,145 mJ to the trunk, 26,260 mJ to the extremities	Reduction of pruritus Flattening of nearly all nodules with a few refractory lesions on the extremities	2.5 months
4	60 years	Trunk, extremities, and scalp	Severe	30+ years	Unknown	Clobetasol 0.05% ointment, narrowband UVB	29 days	105 mJ to the scalp, trunk, and extremities	2% coal tar, clobetasol 0.05% ointment to the trunk and extremities, clobetasol 0.05% solution to the scalp	1+ erythema	3480 mJ to the scalp, trunk, and extremities	Resolution of pruritus Flattening of the majority of nodules	N/A

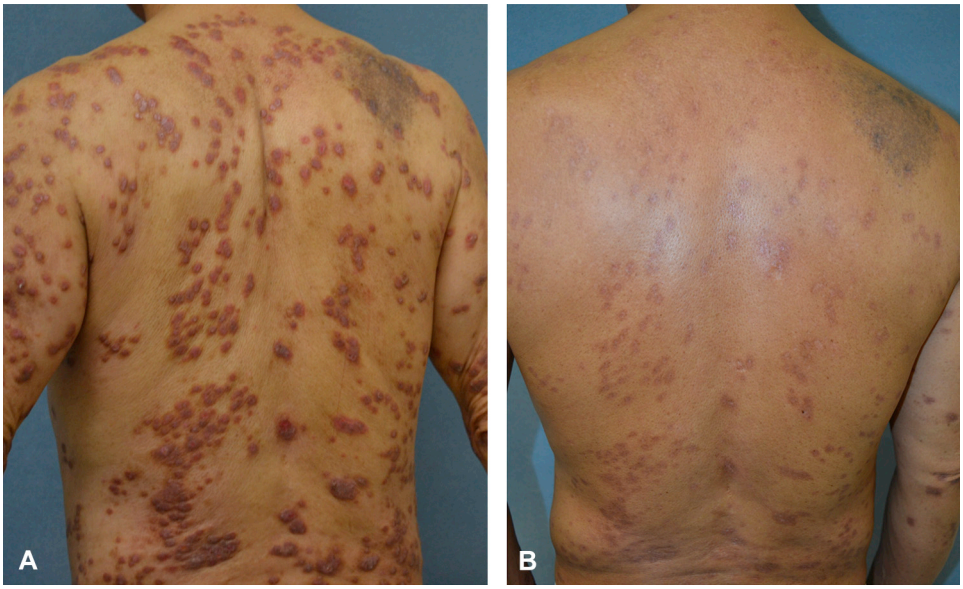

Fig 1. Generalized prurigo nodularis. Patient 1 before (**A**) and after (**B**) a course of modified Goeckerman therapy.

Pfizer, and Merck. Dr Berger is a consultant for Hyperion Therapeutics and Prescription Solutions. Mr Sorenson and Dr Levin have no conflicts of interest to declare.

Correspondence to: Eric Sorenson, AB, 515 Spruce Street, San Francisco, CA 94118

E-mail: esorenso@usc.edu

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PsAPASH: A new syndrome associated with hidradenitis suppurativa with response to tumor necrosis factor inhibition

To the Editor: A 50-year-old man with refractory and multitherapy-resistant hidradenitis suppurativa was referred for evaluation after having been unsuccessfully treated with dapsone, oral isotretinoin, and several cycles of antibiotics (clindamycin-rifampin, tetracycline, cephalosporin) (Fig 1, *A*). He had hidradenitis suppurativa since the age of 43 years, was overweight and a heavy tobacco smoker, and had a medical history of acne, diabetes mellitus type 2, arterial hypertension, hypertriglyceridemia, hiatal hernia, depression, and psoriatic arthritis (PsA). Active acne pustules and comedonal lesions were observed on the face and neck; painful sterile abscesses and hypertrophic scars were present at the axillae and were classified as hidradenitis suppurativa Hurley II stage of severity. The patient had also erythematous scaly lesions on the scalp associated with severe joint and diffuse inflammation leading to the clinical diagnosis of PsA (Psoriasis Area Severity Index score 1.2; DAS28-CRP4 5.78; pain visual analog scale score 70). Two ulcerative lesions on his right leg had a dusky erythematous undermined edge (Fig 1, *B*), and were clinically and histologically diagnosed as pyoderma gangrenosum after the exclusion of diagnoses including neutrophilic disorders, vasculopathies, and infections. His quality of life was severely hampered by disability and social discomfort.

Adalimumab is a highly specific tumor necrosis factor (TNF)-alfa inhibitor, binding to both soluble